Perceptual and Motor Skills, 1994, 78, 999-1009. © Perceptual and Motor Skills 1994

THE SENSED PRESENCE AS RIGHT HEMISPHERIC INTRUSIONS INTO THE LEFT HEMISPHERIC AWARENESS OF SELF: AN ILLUSTRATIVE CASE STUDY[1]

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Summary.—The hypothesis of vectorial hemisphericity predicts that left hemispheric intrusions of the right hemispheric equivalent of the sense of self should be associated with the experience of a "presence" of someone else. The neurophenomenological profile of a woman whose medical history satisfied these theoretical criteria (verified electrical anomalies that could encourage phasic discharges within the right temporal lobe and atrophy within the left temporoparietal region) is presented. In addition to interactions between electrical seizures and thinking, she reported a long history of sensed presences, ego-alien intrusions, and "sudden knowing of the subsequent sequences of seizures" before they occurred clinically. The existence of these neurocognitive processes demands a reevaluation of the psychiatric default explanations of "hysteria" and questions the belief that "awareness during seizures" or "premonition of subsequent somatosensory experience" contraindicates an epileptic process.

Isolation of the neurocognitive substrates for the sense of self will be the major problem for neuropsychology in the twenty-first century. The solution will require a synthesis of phenomenology with the intricate details of neurofunction. Although some traditional neuropsychologists and neurologists have ignored or demeaned the possibility that systematic evaluations of subjective experiences could be employed as inferences of brain function and its dynamic organization (Pawlik & Weiss, 1989), there is strong evidence that those portions of the brain that are most metabolically active at any interval during psychological time determine both the content and the themes of experiential reports (Andreasen, 1988).

Several researchers (Edelman, 1989; Jaynes, 1976; MacLean, 1990) have argued that consciousness emerges from the comparator operations between limbic (affective) and cortical (perceptual) domains and that the sense of self and awareness are secondary to the development of microsynaptic changes that result in the emergence of linguistic functions within the (normal) left hemisphere (Corina, Vaid, & Bellugi, 1992). We (Persinger & Makarec, 1987, 1991, 1992) have hypothesized that the sensed presence (a feeling someone

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or something is present, usually along the back left side) is due to transient interhemispheric intrusions of the right hemispheric equivalent of the sense of self into the left hemisphere.

According to the concept of vectorial hemisphericity (Persinger, 1992a, 1992b, 1993, in press) any condition that facilitates the intercalation of hemispheric operations would increase the likelihood of these experiences. Consequently, populations who exhibit less hemispheric lateralization such as the normal female brain and the left-handed male brain or populations who exhibit phasic increases in hemispheric metabolism such as temporal lobe epileptics and multiple personalities should and do report more of these experiences (Ross, 1989). We now report a unique case in which the phenomenological correlates of these putative intrusions that were characteristic of auras for complex partial seizures were experienced and recorded by the patient.

Clinical Information

Documentation involved access to *all* technical (medical) records and multiple interviews with the patient, her immediate family, and parents. Except for multiple hot baths that were taken by the mother (between 5 and 9 months postconception), the patient's pregnancy and parturition (20 min., no forceps) were uneventful. As a child, the patient was considered very "mature" but fearful; she was easily distressed in open spaces. Except for dysmenorrhea (average duration of menstruation but periods every 1 to 3 months) since menarche, the history of this bilingual, right-lateralized (foot, eye, hand) Roman Catholic woman was unremarkable.

She had been married for three years (no children) and had finished teacher's college (majoring in languages) before the first seizure occurred (age 21 yr.). During the previous 1.5 years she had taught grade school in a different city; she was required to teach in several schools and stress was considered significant. The patient and her husband returned to her parents' home for Christmas holidays. She felt as if she "were in a different world" and displayed "twitching" responses while attending a party. During the subsequent week she developed a fever, sinusitis, and frontal headaches; the left neck was so swollen that the mother suggested mumps. She was ready to attend a midnight mass when she received a distressing telephone call (from a female who was strongly disliked) concerning her brother. Later, she fell backwards into a large snowdrift and was taken to the hospital.

Upon admission this 115-lb. woman's abdomen was so distended that the attendants told her husband that she was in labour (however, she was not pregnant); there were generalized twitching motions. When she awoke after about one week of coma and occasional tonic-clonic convulsions (with and without incontinence and tongue biting), she experienced retrograde amnesia of about three years; she could only recall her parents, sisters (but not the brother), her social security number, and a third language that she had

learned in high school. Discussion of the incidents that led to the initial episode suggested it was associated with generalized myoclonic activity and "twitching."

Subsequent to that period (and for over a decade) she has experienced three types of seizures that have been reduced but not eliminated by all acceptable combinations of anticonvulsant medications. The first (most common) spell involves a sensation of floating and fear; the second involves a period of receptive (auditory) dysphasia. She "knows" when a seizure is going to occur (no auditory correlate) and can terminate ongoing activity, e.g., cooking, in order to sit (average time about 20 seconds). The general aura is experienced as if "*someone* is coming into her mind and directing the seizure"; she feels as if she knows which appendage will be affected. Seizures have occurred in clusters, with no obvious displays for several months, and usually follow the end of menses by about two days; there has been significant premenstrual abdominal swelling and (recently) phasic edema of the right gastrocnemius. Occasional generalization to tonic-clonic convulsions has been reported.

A CT (with contrast) completed about 10 days after the first episode was reported by radiologists to exhibit indications of greater uptake (presumably more parenchymal penetration) in the left posterior hemisphere than in the right. Although the EEG was anomalous it was interpreted by a neurologist to suggest encephalitis rather than epilepsy; however, the cerebrospinal fluid was normal. About 1.5 years later she was thoroughly examined at a world-class neurological institute. CTs indicated hypoplasia of the left temporal lobe; there was enlargement of the right temporal horn of the lateral ventricle and the right medial fossa was larger than the left. Several sleep EEGs indicated diffuse, intermittent seizure activity primarily from the right temporal lobe with involvement of the adjacent uncinate and parasaggital region and occasionally of the *left* temporal lobe. Since the initial onset she had gained 80 lb. and had given birth to a child.

The neurological examination suggested chronic disease of the right maxillary antrum, asymmetry of the face, mild nystagmus during lateral gaze, and a mildly pale optic disc; she was experiencing episodes of strange odors through the right nostril and soreness of the right side of the throat. Major attacks were sometimes preceded by a hot rising sensation in her right ear and were associated with a left transient Babinski and weakness of the right hand grip and wrist flexion; the head was turned to the right and the eyes were deviated upwards and to the right. There was generalized myoclonus, some facial twitching (followed by left facial weakness), and some postictal dysphasia. The neuropsychological assessment indicated a static lesion of the right temporal lobe; standardized scores for the Wechsler Verbal IQ, Performance IQ (WAIS), and Memory Quotient (Wechsler Memory Scale) were + 1.4, + 0.3, and + 1.9, respectively.

During the subsequent 10 years before the second complete neuropsychological assessment by our group, she gave birth to two more children. Social difficulties, frequent confusion, perceived memory disruption, and enhanced myoclonic seizures, especially during periods of stress, had escalated. A four-year period of decreased bonding (to the children) was associated with about four years of kleptomania. There had been intermittent periods of removing her clothes. Reading had been difficult because the process was experienced as if the spaces between words had been removed. There had been a history of multiple sensed presences. After a seizure-free period of approximately six months, she developed influenza-like symptoms and the seizures returned. Two days after the end of two complete days of assessment (age of 33 yr.), she experienced increased seizure frequency that was now associated with amnesia and automatic behaviors.

Standardized (*z*) scores for her WAIS Verbal IQ, Performance IQ (WAIS—R), and Memory Quotient (Wechsler Memory Scale) were −.17, −0.9, and −0.9, respectively. The Halstead-Reitan Index showed moderate brain impairment. There was severe impairment for the Tactual Performance Test (TPT) scores for left, right, and both hands; whereas memory for shapes was normal, localization was severely impaired. Speech Sounds and Seashore Rhythm tests were normal while dichotic listening (for either and both ears) was severely impaired. Although the Trail Making Test A and B were normal, the Category Test was severely impaired. Foot tap and finger tap were normal bilaterally as was finger agnosia and graphaesthesia. However, toe agnosia and toe graphaesthesia (Richards & Persinger, 1992) were moderately impaired. Although the Hooper Visual Organization and Rey Ostereith Memory (+30 min.) tests were moderately impaired, the drawing (copy) of the latter was normal. There was no evidence of a short-term memory deficit for serial verbal learning.

Except for a sluggish right pupillary response to light, classic visual and oculomotor reflexes were intact; there were no gross parietal lobe or cerebellar indicators. Her valid MMPI profile indicated significant elevations (*T* score of >70) on all the clinical scales except Schizoid, Social introversion, and Androgyny; the largest elevations (*T*>90) occurred for Paranoia and Hypomania. Her Complex Partial Epileptic-like Signs score (Roberts, Varney, Hulbert, Paulsen, Richardson, Springer, Shepard, Swan, Legrand, Harvey, & Struchen, 1990) was 4.6 standard deviations above the mean. A screening EEG (Persinger, 1992a; it employs traditional F7, F8, T3, T4, O1, O2 positions) indicated episodic spikes over the temporal lobes; however, the most anomalous observation was the conspicuous weakness of interhemispheric coherence of spindles during left-right frontotemporal comparisons. [These patterns were not considered significant by a neurologist who employs qualitative (visual) criteria but were within the magnitude that is discriminated by QEEG or quantitative electroencephalographic analyses.]

Conclusions and Interpretations

Human experiences are assumed to be generated by the activity of neurons (not their absence). We hypothesize that subclinical complex partial seizures, with occasional overt and traditional displays, were initiated in this woman's brain by a viral infection that compromised the blood-brain barrier; as groups of neurons became active, dominated the phenomenology, and then died from excitotoxic consequences (Bureau & Persinger, 1991; Persinger, Bureau, Kostakos, Peredery, & Falter, 1993), the specific behavioral manifestations changed. The slow and gradual loss of neurons allowed the protracted experience of phenomenological states and psychological experiences that would have been too brief to discern or to discriminate had there been sudden and conspicuous neuronal dropout.

Because subjective experiences, overt behavior, and even the sense of self are assumed to be manifestations of neuroelectrical patterns within the brain, *subtle* but anomalous activation of clusters of neurons within the temporal lobes should first affect phenomenology, then personality, and ultimately cognitive capacity. There is growing evidence for an epileptic spectrum disorder (Roberts, Gorman, Lee, Hines, Richardson, Riggle, & Varney, 1992) with which is associated all of the phenomenological and psychiatric symptoms that were exhibited by this woman. This spectrum appears to be the more extreme component of the continuum of temporal lobe lability (Persinger & Makarec, 1987, 1993).

The likelihood of a congenital or ontogenetically acquired anomaly in the right amygdaloid-parahippocampal region was indicated by the morphological asymmetries of the skull and temporal ventricle and the pattern of the dysmenorrhea. A predominance of right hemispheric electrical foci has been documented in temporal lobe epileptic women who exhibit intrusive sexual ideation (Remillard, Andermann, Testa, Gloor, Aube, Martin, Feindel, Guberman, & Simpson, 1983) and reproductive endocrine anomalies (Herzog, Seibel, Schomer, Vaitukaitis, & Geschwind, 1986) [in fact, subjective pseudocyesis and irregular menstruation are associated with elevated right but not left hemispheric indicators in normal university women (Persinger, 1991)]. The patient's "early maturity" and fear (or "hypervigilance"), which could have met the criterion of a childhood avoidance disorder, implies an early anomalous elevation in amygdaloid activity. The specific association between visuospatial contexts (open spaces) and apprehension suggests a right hemispheric predominance.

Because right amygdaloid activity is often associated with anger, the agitating information about the patient's brother may have evoked such intense stimulation of the central amygdaloid nuclei (Liang, Melia, Campeau, Falls, Miserendino, & Davis, 1992) that the correlative release of the powerful endogenous epileptogenic neuropeptide CRF (corticotrophin releasing factor)

may have precipitated the major event that led to hospitalization. CRF has been shown to increase the spontaneous discharge frequency of CA1 and CA3 pyramidal neurons of the hippocampus by decreasing the size of the normal postburst hyperpolarization (Aldenhoff, Gruol, Rivier, Vale, & Siggins, 1983).

That stress-induced enhancement of central cholinergic activity (Dilsaver, 1988) facilitates release of CRF (Stokes & Sikes, 1988) and subsequent elevation of ACTH (as well as beta-endorphin and cortisol) is clear (Risch, Kalin, Janowsky, Cohen, Pickar, & Murphy, 1983). The aberrant amygdaloid-hypothalamic activity would have stimulated gastric secretion (and stomach distention) and the release of vasopressin and abdominal fluid retention.

Either the fall or correlative diffuse microhemorrhaging (suggested from the first CT, with contrast) from hypertension [perhaps even exacerbated by a viral infection, which if accompanied by the normal elevation of interleukin would have released CRF (Ohgo, Nakatsuru, Oki, Ishikawa, & Matsukura, 1991)] within the left temporoparietal region resulted in a microstructural change that allowed subsequent frequent intercalation with the homologous right temporoparietal region. Encephalitis secondary to chicken pox (varicella) is frequent in adults; considering her vocation (grade school teacher), the time of year, and the likely immunosuppression (from schedule distress), she could have been exposed to the virus. The anomalous swelling along the left parotid region would have been compatible with inflammation and necrosis within the left (temporal) hemisphere (as determined by CT) if a viral/bacterial infection had penetrated the middle ear and reflux nasal mucous had been repeatedly passed through the eustachian tube.

The different types of seizures are reflective of the relative degree of spread of paroxysmal activity between the right and left hemispheres. As shown by nocturnal EEGs, the anomalous electrical activity was almost always within the right temporal lobe and adjacent limbic lobe. If the major epileptogenic area was within the right amygdaloid-parahippocampal region, then fear and floating experiences (similar to panic attacks) would be expected as the paroxysmal discharges accessed the left hemisphere through the anterior commissure. Because this region is the likely electrical source, these types of attacks should have been (and were) the most frequent. Considering the role of the right hemisphere in mediating visuospatial relationships and the amygdala's contribution to aggressive ideation, the "trigger" for these attacks as well as limbic myoclonic displays during periods of social interaction would be expected; emotional triggers for complex seizures have been established (Feldman & Paul, 1976).

The phenomenology of the second type of seizure is most relevant to the hypothesis. If the right hemispheric paroxysmal activity spread beyond the mesiobasal structures and accessed the right temporoparietal lobe, then

discharges to the left homologous areas through the corpus callosum would occur. The intrusion of the right hemispheric homologue of the left hemispheric sense of self was experienced as "another mind entering hers." The "feeling of somebody being nearby" has been associated with surgical and spontaneous stimulation of right temporal lobe activity (Ardila & Gomez, 1988).

Because the actual sequential pattern of discharge from the limbic motor areas would be reflected in the information propagated from the right to the left temporoparietal region [where she would be aware of it, using Edelman's (1989) primary to secondary consciousness model], she would experience the "premonitional sequence" of "knowing which arm or leg would be affected." The mediation of this information from the right to the left hemisphere would be a neuropsychological correlate of the "hidden observer" phenomenon that is evident during hypnosis (Persinger, 1993, in press).

The time-frame of the "premonitional" experience would be expected to exceed 10 seconds (Bossi, Munari, Stoffels, Bonis, Bacia, Talairach, & Bancaud, 1984). Recently, Wilson, Isokawa, Babb, Crandall, Levesque, and Engel (1991) have emphasized that electrical stimulation of the temporal lobes of complex partial epileptic patients evokes widespread paroxysmal displays ipsilaterally; contralateral propagation which often leads to marked alterations in consciousness is very rare. In fact, spontaneous focal seizures may continue for as long as one or two minutes (Wilson, *et al.*, 1991) before they either propagate to adjacent or contralateral structures or fade into background activity.

If the discharge patterns from the right temporofrontal region descended through the central amygdala to brain-stem pathways to access the left hemisphere, at least 20 seconds (Lieb, Hoque, Skomer, & Song, 1987) could elapse. This duration would be sufficient for her to "sit down and wait for the seizure to occur." Because the female brain may be less electrically lateralized (more coherent; e.g., Corsi-Cabrera, Herrera, & Malvido, 1989) and she was multilingual, one would have expected more language representation within the right hemisphere and hence greater fidelity (accuracy) between the two hemispheres in the translation for the imminent sequences of motor activity (Persinger, 1993, in press).

The occurrence of these intrusions from the right temporal lobe foci would have been facilitated by the neuronal dropout within the left temporoparietal region of inhibitory interneurons (Sloviter, 1987) that would have normally prevented the intercalation, i.e., maintained the electrophysiological phase-shifted digital patterns between the two hemispheres. When the neuronal degeneration was sufficient to induce resonance-like paroxysmal patterns in the left hemisphere (bilateral involvement), she began to lose consciousness and to experience periods of disorientation and amnesia.

The emergence of "kleptomania" for about four years, during which time the patient felt "compelled" or was "told" to take objects, would be considered a variant of the ego-alien intrusion experiences that would be associated with transient right hemispheric input and "forced thinking" [psychopathy, hysteria, and 'schizophrenic impression,' i.e., schizophreniform displays, have been frequent descriptors for patients with temporal lobe epilepsy (Bingley, 1958)]. A clinical interview supported the prediction that these events followed periods of anger and social conflict when enhanced lability of the right amygdala would have been expected. Sudden right hemispheric intrusions during periods of low left temporofrontal activity (psychological depression) when the sense of self was weakly integrated (which in our model is simply a function of the relative metabolic activity of this region) would have generated aggression towards the self, i.e., suicidal ideation.

Sexual automatisms, defined as clinical mannerisms that encompass exhibitionism, masturbatory activity, or other sexual activity for which the patient is amnesic, involve frontal lobe anomalies (Spencer, Spencer, Williamson, & Mattson, 1983). In fact, patients with complex partial seizures [which involve foci within the sagittal fissure or orbital area of the frontal lobes] display *unstereotyped* automatisms that react to external stimuli, are not associated with loss of awareness or postictal confusion, and occur in clusters (Waterman, Purves, Kosaka, Strauss, & Wada, 1987). The involvement of the medial frontal surface and supplementary motor area in the seizure pattern of this patient (from restless legs to neuropsychological impairment) suggests this process.

The third major class of seizures which characterized the clinical manifestations would occur less frequently and only after the paroxysmal activity spread from the other regions into the motor cortices. The left temporoparietal region was the source of the clinical seizures (right-side manifestation) and, because the foci were large, was associated with neuronal dropout. That an insidious process had been occurring was indicated by the precipitous drop in Verbal IQ (more than 3.0 standard deviations) relative to the Performance IQ (about 1.0 standard deviation) over a 10-year period. The amount of cell loss that would be required to produce this deficit would be sufficient to form the critical number of foci from which clinical seizures could be generated.

Not surprisingly, given the phenomenological patterns this woman reported, her problems and experiential reports were often dismissed by friends, family, and several attending physicians as "hysterical" or "psychiatric" because there was (1) the triggering by social events, (2) "awareness of the seizure sequence, often in advance," and (3) the frequent absence of incontinence during the myotonic and tonic-clonic displays. Were it not for the expert evaluations by neurologists from a prestigious neurological institute

and the recalcitrance of a young general practitioner, she would have probably been placed in a psychiatric unit or given classic neuroleptic drugs [that would have hastened the degenerative seizure process (Bureau & Persinger, 1991)] long before our assessment was requested.

The integration of her medical history with our neuropsychological assessment and the current model suggests that "awareness of imminent involuntary behavior," the sensed presence of ego-alien intrusions, and classic Schneiderian signs (Ross, 1989) may occur when the right hemispheric equivalent of the self penetrates into the left hemispheric and conscious self. Serious documentation and appreciation for the patient's experiential phenomena rather than abject dismissal as "psychological noise" may aid identification of neurocognitive structures of awareness and sense of self. Isolation of the mediating neuropathways that are correlative with the experiential phenomena, especially when alterations in the spatial distribution of discharges are evident, may facilitate more effective pharmacological or neurocognitive interventions.

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Accepted March 14, 1994.